REMARKS

Claims 6-8 are pending. The arguments presented in the Response filed on June 16, 2009 are incorporated by reference.

Claim Rejections -- 35 U.S.C. 103

Applicants respectfully traverse the anticipatory rejection of claims 6-8 as allegedly anticipated by Chang (Acta Pharmacologica Sinica, August 2003, 24: 796-804). The Examiner relied on Chang for teaching the use of L-butylphthalide to improve ischemia-induced apoptosis. Without acquiescence with the rejection, applicants have deleted "preventing" from claim 6 and thus, rendering the anticipatory rejection moot. The prior art reference does not teach using L-butylphthalide to treat cerebral infarct in human beings by reducing the volume of the cerebral infarct. Chang is silent on the volume of the cerebral infarct. Withdrawal of the rejection is requested.

Claim Rejections -- 35 U.S.C. 103

Applicants respectfully traverse the rejections of claims 6-8 as allegedly obvious over Chang.

Chang discloses that L-butylphthalide inhibited transient focal cerebral ischemia-induced apoptosis (see the first sentence of the Discussion section, p. 802). Chang discloses that L-butylphthalide could reduce the release of cytochrome C, decrease the activation of caspase-3 and inhibit DNA fragmentation after focal cerebral ischemia. However, Chang does not teach or suggest using L-butylphthalide to reduce the volume of cerebral infarct. Chang did not measure the size of the cerebral infarct. Chang is silent on the volume or size of the cerebral infarct. Merely inhibiting transient cerebral ischemia-induced apoptosis does not offer a reasonable of success that L-butylphthalide can treat cerebral infarct by reducing the volume of the cerebral infarct. The relationship of neuronal apoptosis and infarct size is complicated. It was reported that the change of neuronal apoptosis was correlated to the infarct size (Chang et al., J. Biomed. Sci. 2009, 16:9, which was published before the effective filing date of the instant patent application). However, Wang et al. demonstrated that TACE inhibitor reduced infarct size neurological deficits, and had no effect on apoptosis measured by levels of active caspase-3

expression and DNA fragmentation (Wang et al., *Inhibition of tumor necrosis factor-alpha-converting enzyme by a selective antagonist protects brain from focal ischemic injury in rats,* Mol. Pharmacol. 2004; 65(4):890-6, a copy of which is enclosed). Thus, to a person of ordinary skill in the art, there is no reasonable expectation that L-butylphthalide would reduce the volume of the cerebral infarct, based merely on Chang's disclosure that L-butylphthalide inhibited transient focal cerebral ischemia-induced apoptosis the inhibition of apotosis. Without a reasonable expectation of success, Chang does not render obvious the claims 6-8. At least due to the reasons discussed above, withdrawal of the obviousness rejection is requested.

The Examiner is invited to contact the undersigned at 202-220-4200 to discuss any matter in connection with this application.

In the event that the filing of this paper is deemed not timely, applicants petition for an appropriate extension of time. The Office is hereby authorized to charge any fees in relation to this paper under 37 C.F.R. 1.16 and 1.17 to the Kenyon & Kenyon Deposit Account No. 11-0600.

Respectfully submitted,

Dated: June 29, 2009

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Enclosure: Wang et al., Mol. Pharmacol. 2004; 65(4):890-6.

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